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Family and genetic group effects for resistance to proliferative gill disease in channel catfish, blue catfish and channel catfish × blue catfish backcross hybrids

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Abstract

The severity of gill damage (percentage of gill lamellae with lytic lesions) was determined in juveniles from 10 USDA 103 line channel catfish *Ictalurus punctatus* full-sib families, 10 channel catfish × blue catfish *I. furcatus* backcross hybrid (7/8 channel catfish, 1/8 blue catfish) full-sib families and a mixed-family group of blue catfish placed in a commercial catfish pond experiencing proliferative gill disease (PGD)-related fish mortalities. An initial challenge was conducted with all families, and a second challenge was conducted using the two most susceptible (most gill damage) channel catfish and backcross hybrid families and the two most resistant (least gill damage) channel catfish and backcross hybrid families. In the initial challenge, percentage gill damage was not different between channel catfish (12.3%) and backcross hybrids (11.6%), but was lower in blue catfish (0.2%). Mean percentage gill damage in the second challenge was not different among resistant backcross hybrid families (6.9%), resistant channel catfish families (7.6%) and blue catfish (4.8%), but was higher in susceptible backcross hybrid and channel catfish families (19.0% and 11.9% respectively). The correlation among family means for gill damage from challenge 1 and challenge 2 was $r = 0.87$. Consistent differences between channel catfish and blue catfish and between resistant and susceptible families within genetic groups for gill damage after PGD challenge suggest that there is a genetic component for resistance to PGD and that improving PGD resistance through selection may be possible.

Keywords: catfish, proliferative gill disease, resistance

Introduction

Proliferative gill disease (PGD) is a major disease problem of farm-raised catfish and can result in mortality rates of > 50% in severe cases (Bowser, Munson, Jarboe & Stiles 1985). A recent survey of 61 commercial catfish ponds found that some fish from all ponds showed signs of PGD-related gill damage at some point during a 4-month period (Wise, Terhune & Khoo 1999). The disease is characterized by inflammation and damage of gill tissue resulting in impaired osmoregulation and oxygen transfer (MacMillan, Wilson & Thiyagarajah 1989). Although the life cycle of the disease-causing organism is not completely understood, molecular genetic data and histopathology of the disease suggest that the causative agent, identified as *Aurantiactinomyxon ictaluri*, is the actinosporean stage of the myxospore *Henneguya ictaluri* (Pote, Hanson & Shivaji 2000; Hanson, Lin, Pote & Shivaji 2001). Research is being conducted to determine management and treatment strategies to reduce PGD-related losses, but there is currently no effective means of treating PGD.

Treatment of fish diseases is frequently complicated because of limited availability and effectiveness of therapeutics (Chevassus & Dorson 1990). Therefore, genetic selection of cultured fish for improved disease resistance may be an effective approach to

reducing economic losses from diseases. The basis for trait improvement through genetic selection is the use of existing genetic variation for the trait of interest. The genetic variation used as the basis for trait improvement can be variation among genetic groups (species or breeds), among individuals/families within a breed or species or a combination of both. Estimation of the degree and type of genetic variation for a trait can be a complicated and resource-demanding endeavour, but a simple comparison of a trait between various breeds/species or between families within breeds or species can be indicative of the presence of genetic variation.

Past research at our facility (D J Wise, unpubl. data) demonstrated that blue catfish *Ictalurus furcatus* and channel catfish *I. punctatus* × blue catfish F1 hybrids exhibit less gill damage than channel catfish when fish are placed in ponds experiencing PGD-related fish mortalities, and indicates the presence of genetic variation for PGD resistance between blue and channel catfish. However, commercial culture of blue catfish is limited because they possess other unfavourable production characteristics compared with channel catfish (Dunham, Hyde, Masser, Plumb, Smitherman, Perez & Ramboux 1993), and commercial use of channel catfish × blue catfish hybrids is limited by reproductive barriers that hinder hybrid fingerling production. Increasing PGD resistance while maintaining good overall culture characteristics (e.g. spawning performance) may be possible through the development of a synthetic 'breed' derived from channel catfish × blue catfish backcross hybrids. Additional improvements in PGD resistance may be possible by using variation among families, either within a synthetic breed or within purebred channel catfish strains. The objective of this study was to determine the effects of genetic group and family within genetic group on gill damage in channel catfish, blue catfish and channel × blue catfish backcross hybrids exposed to a commercial culture pond with fish experiencing PGD-related mortalities. The results will provide information on the potential for increasing PGD resistance through genetic selection.

Materials and methods

PGD challenge protocol

Juvenile catfish were stocked into nylon mesh, circular net pens (1 m deep × 0.7 m diameter) placed in a commercial catfish pond experiencing PGD-related fish mortalities. PGD diagnosis was based on histological examination (epitheal proliferation, lysis of chondrocytes, fusion of secondary lamellae and presence of parasitic cells identified as *Aurantiactinomyxon ictaluri*; MacMillan *et al.* 1989) of gill tissue and was conducted by the Fish Disease Diagnostic Laboratory, College of Veterinary Medicine, Mississippi State University, Stoneville, MS, USA. After 7 days, fish were removed from net pens, transported back to our laboratory, killed by overdose with tricaine-methyl sulphonate, and wet mounts of 30–80 gill filaments per fish were prepared. The percentage of primary gill lamellae containing lytic lesions was determined by microscopic examination of gill filaments, and percentage gill damage was used to quantify the presence and severity of PGD for each fish. The severity of gill damage determined by microscopic examination of wet mounts is positively correlated with PGD-associated histopathology and is a good early indicator of subsequent levels of PGD-related mortalities in commercial ponds (D J Wise, unpubl. data). A random sample of three prechallenge fish from each genetic group and three post-challenge fish from each genetic group in challenge 1 was examined by personnel from the College of Veterinary Medicine, Mississppi State University, for histopathology (see above) indicative of PGD.

Challenges

Fish used in challenges were hatched and reared in indoor tanks supplied with well water (26 °C) at the Catfish Genetics Research Unit, USDA-ARS, Stoneville, MS, USA, before challenges. Fish were fed the same diets and were approximately the same age (9–10 months old) at the time of challenge. An initial PGD challenge was made with juveniles from 10 USDA 103 line channel catfish full-sib families (mean weight 65 g), 10 channel catfish × blue catfish backcross hybrid (7/8 channel, 1/8 blue catfish) full-sib families (91.6 g) and a mixed-family group of blue catfish (40.6 g). Each channel catfish family and each channel catfish × blue catfish backcross hybrid family was stocked in a separate net pen (12 fish per net pen, total of 20 net pens), and blue catfish were stocked in six replicate net pens (12 fish per net pen) in a pond experiencing PGD-related fish mortalities. After 7 days, all surviving fish were removed, and gill damage was quantified as described above.

Data from the first challenge were used to identify the two most susceptible (most gill damage) and least susceptible (least gill damage) channel catfish and

backcross hybrid families for use in a second challenge. A second challenge was conducted using the four susceptible and four resistant families (five replicate net pens per family, 10–12 fish per net pen) and six replicate net pens of mixed-family blue catfish fingerlings (12 fish per net pen). Surviving fish were harvested after 7 days, and percentage gill damage was determined. The second challenge was started 2 weeks after the first challenge ended.

Data analysis

A one-way ANOVA was used to compare fixed effects of genetic group for percentage gill damage, survival and weight in the first challenge. A two-way ANOVA was used in the second challenge and included fixed effects of genetic group, resistance group (resistant vs. susceptible) and their interaction. Mean × square e errors associated with replicate cage within genetic group and replicate cage within genetic group × resistance group were used as error terms in tests of significance in the first and second challenges respectively. Pearson product–moment correlations between family means for percentage gill damage in the first and second challenges were determined. Statistical analysis was conducted using SAS (SAS 1988). Differences were declared significant at $P < 0.05$ unless otherwise noted.

Results

Challenge 1

Prechallenge fish (three per genetic group) did not exhibit histology associated with PGD (breaks in lamellae, epitheal proliferation, lysis of chondrocytes, fusion of secondary lamellae and presence of parasitic cells identified as *Aurantiactinomyxon* sp.). Histology of post-challenge fish was indicative of PGD, including the presence of parasitic cells. The limited sample size did not allow statistical comparison of histopathology of genetic groups, but a subjective assessment indicated that these characteristics were more prevalent in gill tissue from channel catfish and backcross hybrids than in gill tissue from blue catfish.

Mean percentage gill damage was not different between channel catfish (12.3%) and backcross hybrids (11.6%), but was lower in blue catfish (0.2%) (Table 1). Mean percentage gill damage for families ranged from 5.3% to 20.6% in channel catfish and from 5.7% to 26.9% in backcross hybrids. Survival during the challenge was similar in channel catfish (98%) and channel catfish × blue catfish backcross hybrids (97.5%) and lower in blue catfish (70%). Backcross hybrids (mean weight 91.6 g) were larger than channel catfish (65.0 g), and both groups were larger than the blue catfish (40.6 g). Within channel catfish and backcross hybrids groups, correlations among family means for weight and percentage gill damage were not different from zero.

Challenge 2

Mean percentage gill damage was not different among resistant channel catfish families (7.6%) and resistant backcross hybrid families (6.9%) and blue catfish (4.8%), but was higher in susceptible channel catfish families and backcross hybrid families (11.9 and 19.0% respectively) (Table 2). The correlation among family means for gill damage from challenge 1 and challenge 2 was 0.87 ($P = 0.005$). Survival (resistant and susceptible families combined) during the second challenge was highest in channel catfish (94.2%), intermediate in backcross hybrids (78.4%) and lowest in blue catfish (66.7%). Survival was not different between resistant and susceptible channel catfish, but was higher for resistant backcross

Table 1 Challenge 1: mean weight, percentage gill damage and survival of channel catfish, channel catfish × blue catfish hybrids and blue catfish*

	Channel catfish	Backcross hybrids	Blue catfish	SE
Weight (g)	65.0[a]	91.6[b]	40.6[c]	6.3
Gill damage (%)	12.3[a]	11.6[a]	0.2[b]	1.8
(range of family means)	5.3–20.6	5.7–26.9	0–0.9	
Survival percentage	98[a]	97.5[a]	70.0[b]	3.7

*Within a row, values with a different superscript letter are significantly different at $P < 0.05$.

Table 2 Challenge 2: percentage gill damage and survival of mixed-family blue catfish and resistant and susceptible channel catfish and channel catfish × blue catfish backcross hybrid families*

	Resistant		Susceptible			
	Channel catfish	Backcross hybrids	Channel catfish	Backcross hybrids	Blue catfish	SE
Weight g	55.0[b]	91.5[a]	58.0[b]	81.5[a]	39.8[c]	6.1
Gill damage (%)	7.6[a]	6.9[a]	11.9[b]	19.0[c]	4.8[a]	1.4
Survival	96.7[a]	81.7[b]	93.3[a]	68.3[c]	70.8[c]	4.6

*Within a row, values with a different superscript letter are significantly different at $P < 0.05$.

hybrids (81.7%) than for susceptible backcross hybrids (68.3). Backcross hybrids (mean weight 86.5 g) were larger than channel catfish (56.5 g), and both groups were larger than the blue catfish (39.8 g), but weight was not different among resistant and susceptible fish within a genetic group.

Discussion

Differences between channel catfish and blue catfish and between resistant and susceptible families within channel catfish and backcross hybrids for gill damage after PGD challenge suggest the presence of a genetic component for resistance to PGD. Our present study confirms earlier, unpublished reports that blue catfish are generally less susceptible to PGD infections than channel catfish. Hedrick, McDowell, Gay, Marty, Georgiadis & MacConnell (1999) reported differences between brown and rainbow trout for resistance to whirling disease, a fish disease caused by another myxosporean (*Myxobolus cerebalis*). El-Matbouli, Hoffman, Schoel, McDowell & Hedrick (1999) demonstrated that *M. cerebalis* has host specificity for salmonids and that this host specificity results from both mechano- and chemotactic stimuli. The differences in PGD resistance among channel catfish and blue catfish suggests that *A. ictaluri* also demonstrates some degree of host specificity. Differences in PGD resistance among catfish species and among families within genetic groups provide an opportunity to identify the basis of host specificity of *A. ictaluri* and could provide information useful for increasing PGD resistance through genetic selection.

Although blue catfish had less gill damage than channel catfish, there was no difference in gill damage between channel catfish and backcross hybrids. The lack of difference in gill damage between channel catfish and backcross hybrids may be related to the fairly high percentage of the channel catfish genome (theoretical average = 7/8) of the backcross fish used in this study. More information on the relationship between percentage of blue catfish genome and resistance to PGD could be obtained by including hybrids with a greater percentage of blue catfish genome (e.g. F1: 1/2 channel, 1/2 blue catfish) in challenges.

To avoid bias in estimates of gill damage resulting from differential mortality, we attempt to end PGD challenges before the onset of significant PGD-related mortalities. However, significant mortalities were observed in blue catfish before the termination of both challenges. We do not believe that the mortalities observed in the blue catfish were related to PGD as most of the blue catfish mortalities occurred early in the study (within 48 h after stocking), and surviving blue catfish had less gill damage than channel catfish and backcross hybrids. Typically, with the PGD challenge protocol used, it takes several days before mortalities occur, and the level of mortality increases as the level of gill damage increases (D J Wise, unpubl. data). Therefore, it seems likely that blue catfish mortalities were primarily related to transport and stocking stress and not to PGD. Blue catfish are less tolerant of transport and handling stress than channel catfish (Dunham *et al.* 1993). The pattern of mortalities (most mortalities occurring from days 5–7 of the challenge) and the higher degree of gill damage in susceptible backcross families than in resistant backcross families in the second challenge suggests that the lower survival in susceptible relative to resistant backcross families was probably caused by PGD-related mortality.

The high correlation between family means for gill damage between the two challenges suggests that differences in PGD resistance were not based on temporary environmental effects. The consistent family rankings for gill damage and differences among blue catfish and other genetic groups tested suggest a genetic basis for PGD resistance. The

potential for increasing PGD resistance in farm-raised channel catfish through genetic selection should be investigated.

Large-scale trials are needed to quantify the type and magnitude of genetic variation for PGD resistance in catfish. Screening large numbers of families for PGD resistance can be achieved using the challenge protocol and techniques for quantifying gill damage described. The challenge protocol could be used to identify and select the most PGD-resistant families, and these fish could be used as broodstock in a family selection approach to increasing PGD resistance. Future research should focus on screening larger groups of catfish (more lines, more families) and determining the response to selection for PGD resistance in farm-raised catfish.

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